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Dynamic Quadriceps Muscle Stimulation for Treatment of Patellofemoral Pain

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Dynamic Quadriceps Muscle Stimulation for Treatment of Patellofemoral Pain

by

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Dedication

To my grandmother who passed away before the completion of this thesis. “Madarjoon”- without your countless sacrifices in life I would not be the person I am today. I still feel you deep in my heart. You won’t be forgotten.

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Abstract

Dynamic Quadriceps Muscle Stimulation for Treatment of Patellofemoral Pain

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Introduction: Patellofemoral pain syndrome (PFPS) is one of the most common types of chronic knee pain. In order to treat PFPS, electrical stimulation (ES) is widely used. The primary goal of the present study was to investigate whether electrical stimulation of the vastus medialis oblique (VMO) and vastus lateralis (VL) to establish coactivation of these muscles during terminal swing phase of normal walking impacts factors associated with PFPS such as abnormal quadriceps muscle activation. **Methods**: Twelve persons diagnosed with PFPS participated in this study. Each participant completed four trials of normal walking. Each trial lasted for 6 minutes. VICON motion capture system was utilized to record the kinematic pattern of movement. VMO and VL muscles' activity were recorded using mechanomyographic (MMG) technique. In order to electrically stimulate VMO and VL muscles, a wearable stimulator, "KneeStim (Articulate Labs, Inc.)", was utilized. During trial one, participants walked without wearing the stimulator. During trial two, participants wore the KneeStim but the stimulator was off. During trial three, the

stimulator was on at the beginning of each stride for the entire swing phase and electrically stimulated VMO and VL muscles of the symptomatic leg of the participant. Trial four was performed identical to trial two in order to observe the effects of stimulation on the muscles' onset times. Muscle onset times as a measure of muscular activity pattern, stride length and stride period were compared over the entire experiment. **Results**: The onset times of the VMO and VL muscles showed significant change between trial one and trial two, and between trial two and four, but no change when trial one was compared to trial four. The difference of muscles' onset times (VL-VMO) did not change after ES intervention. Stride period did not change, stride length (mm) values slightly decreased for symptomatic leg after participants were exposed to ES. However, this change was significant when non-symptomatic leg was taken into account. **Conclusion**: A single session ES intervention starting at the beginning of each stride showed earlier activation of quadriceps muscles during normal walking in individuals with PFPS. However, the delay onset timing of VL compared to VMO did not change after treatment.

List of Acronyms

PFPS – patellofemoral pain syndrome

ES – electrical stimulation

MMG – mechanomyographic

VMO – vastus medialis oblique

VL – vastus lateralis

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CHAPTER 1

INTRODUCTION

1.1 Background

Patellofemoral pain syndrome (PFPS) is one of the most common forms of chronic knee pain in young and active individuals, particularly in females (Boling et al. 2010). PFPS is an overuse injury causing pain in the anterior side of the knee joint, underneath the patella and on the articular surface of the femur. PFPS has long lasting effects that may linger between 4 and a staggering 18 years after its first initiation in over 90% of the patients (Lankhorst et al., 2012). PFPS patients report that the pain significantly restricts their daily physical activities (Lankhorst et al., 2012). Inaccurate diagnosis and improper treatment of PFPS can lead to a decrease in quality of life, increase in injury risk factors during sport activities or even daily tasks, and more serious chronic pathologic conditions such as osteoarthritis (Kwon et al. 2014).

There are biomechanical and neuromuscular factors associated with PFPS. Among those, abnormal muscle activation patterns or quadriceps muscle deficit, is one of the most frequently identified neuromuscular factors associated with PFPS. Delayed activation of the vastus medialis oblique (VMO) or a relative activity of VMO compared to vastus lateralis (VL), VMO: VL ratio, have been widely studied in clinical settings and have been a major focus in rehabilitation strategies (Sawatsky et al., 2012 and Miller et al., 1997). In terms of biomechanical factors, more specifically kinematics, PFPS patients show shorter

stride length compared to healthy population (Powers et al. 1997). Moreover, lower knee flexion angle, increased hip adduction and internal rotation are reported for PFPS patients compared to healthy individuals during different dynamic tasks (Weiss and Whatman, 2015; Lankhorst et al., 2012).

In order to record and assess the muscle activity, methods such as surface electromyography (SEMG) and mechanomyography (MMG) have been widely used. However, SEMG signal is not immune from movement artifacts and cross talk effect, and the noise produced during dynamic tasks is considerable (Wittek et al., 2001). As a result, SEMG is more accurate when implemented to assess the muscle activity during isometric contraction rather than concentric and eccentric (dynamic contraction).

MMG amplitude is consistent with EMG data during eccentric/concentric (Beck et al., 2005) and dynamic contractions (Shinohara et al., 1997).

In order to reduce and to treat PFPS, different interventions have been implemented such as electrical stimulation (ES), physical therapy, patellar taping, muscle strengthening and stretching. ES can be used to re-educate the firing pattern of a muscle during execution of different tasks. As an example, after a 15-minute patterned ES of gluteus medius (GMED), VMO and hamstrings muscles, female patients diagnosed with PFPS showed improvement in knee flexion and hip abduction kinematics during the lateral step-down exercise (Glaviano et al. 2015). Moreover, patients with PFPS who received a treatment program consisting of retraining and strengthening of quadriceps in addition to patellar taping showed significant reduction in their pain scores (Crossley et al. 2002). However, the literature on using electrical stimulation for patients with abnormal muscle activation

pattern is still limited and remains inconclusive. Additionally, it is still unknown whether this method would be advantageous for PFPS patients to improve performance during normal daily tasks such as walking.

1.2 Statement of purpose

The primary goal of the present study is to investigate whether electrical stimulation of the VMO and VL to establish coactivation of these muscles during terminal swing phase of normal walking impacts factors associated with PFPS. MMG method was chosen to assess the mechanical activity of the muscle during the dynamic task of walking.

1.3 Hypotheses

In this study, it was hypothesized that following the ES intervention starting after each toe-off(TO) lasting for the entire swing phase of each stride, VL and VMO muscles would activate earlier during each gait cycle. In addition, the difference between the onset times of the two muscles would decrease during the swing phase of walking. Moreover, it was hypothesized that the average stride length of the participants would increase following the treatment.

CHAPTER 2

REVIEW OF LITERATURE

2.1 Introduction

Patellofemoral pain syndrome (PFPS) is one of the most common forms of chronic knee pain in young and active individuals, particularly in females. PFPS is an overuse injury causing pain in the anterior side of the knee joint, more specifically, underside the patella and on the articular surface of the femur. Based on the current scholarly literature, PFPS has long lasting effects that may linger between 4 and a staggering 18 years after its first initiation in over 90% of the patients. Moreover, 36% of the PFPS patients have reported that the pain has significantly restricted their daily physical activities (Lankhorst et al., 2012). Inaccurate diagnosis and improper treatment of PFPS can lead to health issues such as decrease in quality of life, increase in injury risk factors during sport activities or even daily tasks, and more serious chronic pathologic conditions such as osteoarthritis (Carlson et al., 2017). In order to achieve a more accurate diagnosis and treatment of PFPS, a more detailed understanding of the biomechanical and neuromuscular factors associated with this injury is a matter of paramount importance. The primary goal of the present chapter is to review the neuromuscular and biomechanical factors associated with PFPS, and to briefly discuss the most common corresponding rehabilitation methods. How these factors may

contribute to initiation of PFPS and the ways to prevent or remedy the resulting pain are discussed.

2.2 Mechanism of the Patellofemoral Pain

Normal process of knee flexion and extension is involved with movement of the patella via the quadriceps tendon inward the trochlear groove of the femur inferiorly and superiorly respectively (Guney et al., 2016). In normal pattern of movement and during knee flexion and extension, there is no contact between the ridge of the patella which is covered by a layer of soft tissue (located on its posterior surface) and the medial and lateral condyles of the femur. Any shift of the patella towards the sides may cause major issues over time. During different tasks and activities such as walking, running, jumping, squatting, etc. that are involved with knee flexion and extension, maltracking in the movement of the patella can cause pain by overuse. In such situations the contact pressure and frictional forces between the patella and the condyles of the femur increases. This causes the soft tissue to wear off that leads to tissue irritation and pain. Moreover, the increased contact pressure inside the patellofemoral joint will be perceived by sensory neurons. These sensory neurons are located in the articular surface of the bone on the periosteum layer which is enriched by nerves and blood capillaries.

As discussed above, maltracking of the patella is one of the main reasons found which directly contribute to initiation of PFPS and maintaining the pain. In the next part of

this chapter we will focus on the main possible reasons behind the maltracking of the patella.

2.3 Main Causes of Patellar Maltracking

In the literature the main categories of measures that may be causative factors in patella maltracking are introduced as clinically measured static alignments, dynamic malalignments, and abnormal muscle activation (Earl et al. 2005).

2.4 Clinically Measured Static Alignments

The main static alignments measured in studies are Q-angle, navicular drop, standing genu valgum, anterior pelvic tilt, and hamstring flexibility. Among mentioned measures Q-angle or quadriceps angle is most studied in the literature and considered as one of the probable contributing factors to PFPS. Q-angle is defined as the angle between two lines; One from tibial tuberosity towards the midpoint of the patella superiorly and the other starting from anterior superior iliac spine (ASIS) towards the midpoint of patella. Larger Q-angles lead to a situation called genu valgum or knock knee. It has been reported that among individuals with larger genu valgum the tightness of vastus lateralis muscle (VL) is common. Consequently, during performing different activities the patella is pulled laterally by quadriceps tendon and causes patellar maltracking. To investigate the likely contribution of the Q-angle to initiation of PFPS, different studies have been done. One study showed significantly larger Q-angle in PFPS patients in weight bearing position compared to healthy control group (Haim et al. 2006). In another study by Thome et al.

(1995), the authors didn't find any differences in Q-angle measures between PFPS individuals and control group. The measurements were done with 0 and 30 degrees of knee flexion. They claimed that Q-angle may be a contributing factor in maintaining the pain once it occurred, but not the main cause to its initiation. Tightness of quadriceps muscle group and more specifically VL and vastus medialis oblique (VMO) due to increased Q-angle may cause maltracking of the patella. As a result, in active individuals with larger Q-angle who are prone to PFPS it's worthwhile to monitor the muscles flexibility to avoid tightness.

In another study by Kwon et al. (2014), authors found a significant decrease in hamstring muscle group flexibility in individuals with PFPS compared to control group. However, no significant differences were found in navicular drop as a measure for hyperpronation of the feet and static Q-angle between patients and healthy young adults. As reported in the literature and discussed by Kwon and his colleagues, imbalance of agonist muscle affects its relation with the antagonist muscle. In this case, shortening of the hamstrings may cause weakening of the quadriceps muscles and as a result, putting more demand on quadriceps muscle to produce power during knee extension. This increases the contact forces inside the patellofemoral joint and causes pain. Consequently, to avoid pain and initiation of PFPS in injury prone individuals, it is necessary to avoid tightness of hamstrings muscle group by performing proper stretching exercises.

2.5 Dynamic Malalignment

Malalignment that occurs during movement because of poor neuromuscular control of the trunk and lower extremity. Main dynamic malalignment measures introduced in the literature are contralateral pelvic drop, femoral adduction and internal rotation, dynamic Q-angle or genu valgum, tibial internal rotation and hyper pronation(Eversion).

Contralateral pelvic drop is common in female runners and has been associated with PFPS. It is hypothesized that contralateral pelvic drop shifts the body center of mass away from the stance leg; therefore, increases the abduction/adduction moment arm and the load on the stance leg's hip joint. As a result, hip musculature must produce more abduction moment to counteract the excessive adduction moment. This increased demand on the hip musculature can be problematic among individuals with weaker hip muscles. In a study by Willson and Davis, (2007) the authors reported increased contralateral pelvic drop in PFPS patients compared to healthy age and activity matched control group. Increased contralateral pelvic drop can cause stretch and elongation of the ipsilateral iliotibial band (IT band). The resulting IT band tension can increase the lateral force on patella leading to maltracking. However, Noehren et al. (2012) did not find any significant difference in contralateral pelvic drop in PFPS patients compared to control. As the results of the study were contradictory to their primary hypothesis, the authors made an intriguing claim in their discussion. They interpret this contradictory result as a mechanism implemented by patients to compensate for weaker hip abductors by reducing the demand on gluteal muscles. To support this claim, they found a reduced contraletaral trunk lean among their patients.

In the same study, the investigators found a significant increase of tibial internal rotation in PFPS group. Tibia's internal rotation is affiliated with foot eversion (or hyper pronation) in the literature. The authors of this study associated the increased tibial internal rotation to hip internal rotation observed in patients. As a final note on this section, we shall talk about the importance of hip abductors in pain prevention in weight bearing activities such as running, squatting, and single leg jumping. During performing such activities and as a result of increased demand for production of abduction moment at the hip joint, gluteus minmus (GMIN) and gluteus medius (GMED) muscles play an important role. In order to avoid pain at the knee joint due to excessive contaralateral pelvic drop, and consequently, IT band tension, hip internal rotation, tibial internal rotation, and foot eversion, and navicular drop strengthening of the aforementioned muscles should be a major consideration for PFPS prone individuals.

In the following section of this chapter we shall talk about abnormal muscle activation patterns that may contribute to PFPS.

2.6 Abnormal Muscle Activation

According to the literature available on this topic, the abnormal muscle activation pattern has been divided in to two main categories; Onset timing of the muscles and the intensity of muscle activation (Dvir et al., 1991 and Guney et al., 2016).

Onset timing of the muscles during a particular task is widely utilized in the literature to assess muscular function. Another muscle function measure, intensity of the

muscle activation, can be interpreted as the muscle strength. Relative normalized EMG activity of two muscles with similar function during a specific task is a common method of comparing the relative strength of the two muscles. In this method EMG activity of the muscles during a desired dynamic task will be normalized to the maximum voluntary contraction (MVC) of the same muscle during an isometric task. When using this method for PFPS patients, the MVC of the symptomatic leg is either compared to the patient's non-symptomatic leg or to the healthy subject in order to find any strength deficit in quadriceps muscles. As a case in point, Miller and colleagues (1997) examined the relative strength of the VMO and VL muscles in PFPS patients while performing closed kinetic chain exercises. During static lunge with 30o and 70o knee flexion, dynamic step-up/step-down exercise, and a modified wall slide movement, PFPS group showed less activity of VMO compared to VL. This resulted in less relative ratio of VMO:VL activity in individuals with PFPS compared to healthy control group.

On the other hand, in a recent study in 2013 by Toumi et al. the authors alleged that PFPS is not necessarily associated with quadriceps deficit. During squat and isometric strength test maneuver, they found delayed onset of VMO muscle (VMO activation deficit) in 17 PFPS subjects and observed quadriceps strength deficit in only 6 PFPS subjects out of 32 patients recruited for the research study. Moreover, they mentioned that weakness of VMO muscle is not necessarily associated with quadriceps strength deficit. This latter conclusion referred to as functional differences observed in VMO and VL. VMO is identified as a knee stabilizer as it can pull the patella medially during knee extension with

its oblique fibers, while VL is known as the main force producer during knee extension (Besier et al. 2009). In this study they also found that in 17 subjects VMO activation deficit preceded pain and in 7 other subjects the diminished activation of VMO followed their PFPS pain. In patient with precedence of pain over muscle onset timing it is important to decrease pain in order to employ any strengthening program for quadriceps muscles. Otherwise, the initiation of pain may cause inhibition of VMO which is not desired in patients who have patella maltracking due to quadriceps strength deficit.

In another study the authors interestingly did not find significant differences in muscle activation level and onset timing of GMED and gluteus maximus (GMAX) for PFPS patients compared to control group. However, they found a 25% increased activation level of GMED in PFPS individuals (Willson et al. 2011). This increase was not significant due to limited number of subjects so there is a probability that a future study with larger number of subjects would observe significant results. In the aforementioned study the authors also reported altered hip internal rotation and adduction observed in the patients which could not be solely related to deficiency of hip adductor and external rotators. As a result, interventions to improve altered lower body kinematics should not focus on gluteal muscle strengthening alone. As cited in this study, adding visual feedback to lower extremity strengthening program significantly improved the frontal plane kinematics of jumping in subjects (Herman et al. 2009). As another note on evaluation of hip musculature weakness, it seems more reasonable to study muscle activation levels during dynamic tasks with higher demand for this muscle group. As an illustration, GMAX muscle is known as the major hip

external rotator, while GMED is known as a major hip abductor. Consequently, short running distance in laboratory settings which mostly demands hip and knee extension/flexion might not be the proper task to evaluate the function and strength of GMED and GMAX muscles. Activities involved with hip external rotation and abduction are more illustrative on this matter. Similarly, in the study mentioned earlier by Miller et al. the authors concluded that closed kinetic chain activities are not the best evaluator of the VMO weakness as they not preferentially cause recruitment of this muscle in individuals with PFPS. Further conclusion would be the fact that muscle strength deficit is a contributing factor to PFPS but not the one and only factor. Therefore, finding the cause of pain in order to implement the best rehabilitation program is crucial.

In the following sections of this chapter we shall discuss the kinematics and kinetics associated with patellofemoral pain syndrome. Moreover, we shall present some of the main findings of the research studies related to the topic. Then, expand the discussion by investigating the relations between the previously discussed factors associated with pain and altered kinetics and kinematics due to PFPS that observed during different activities.

2.7 Kinematics Associated with PFPS

Most frequently reported kinematic measures in the literature are as follows:

- Patella joint kinematics
- Joint angles such as knee flexion, hip adduction and internal rotation
- Stride length and linear velocity of walking

2.7.1 Patella Joint Kinematics

Patella joint kinematics also has been known as a potential factor associated with the maltracking of the patella in PFPS patients and been extensively studied in the literature. In this regard, MacIntyre et al. (2006) acquired low resonance scans of the patella, femur, and tibia in different loaded knee flexion angles (ranging from-4o and 60o) to identify patellar spin, tilt, and lateral translation among PFPS patients. They only found a significant difference in lateral motion of the patella which was 2.25mm more laterally in PFPS group than the control group. In this study only one third of the subjects with PFPS were diagnosed clinically with patellar maltracking and the results cannot shed light on precedence of the maltracking symptoms to onset of pain and vice versa. This also showed that by examining the patterns of spin, tilt, or lateral translation of the patella alone, clinicians one cannot distinguish patients from healthy individuals. However, the maltracking examination is a useful method to confirm patient's PFPS and will remain as a risk factor that may contribute to onset of pain. These findings suggest that more precise examination of the patella joint must be utilized to diagnose PFPS in individuals with knee pain in order to design the best treatment strategies.

2.7.2 Joint Angles

Generally, research articles found less knee flexion angle, increased hip adduction and internal rotation are reported for PFPS patients compared to healthy individuals during different dynamic tasks (Weiss and Whatman, 2015; Lankhorst et al., 2012). This increased

hip internal rotation and adduction most often occur during landing phase of activities such as running, jumping, etc. This can be associated with weakness of hip musculature leading to diminished ability to produce enough hip abduction moment. In the following sections, we will discuss altered kinetics and more specifically altered joint moments due to PFPS to logically explain altered kinematics in PFPS patients. In another note, it seems reasonable to expect less knee flexion peak in patients suffering from PFPS. As explained earlier, during knee flexion patellar ridge glides into the trochlear groove in between the condyles of the femur inferiorly. Moreover, maltracking of the patella identified as one of the possible causes for increased contact pressure in patellofemoral joint. As a result of increased forces and pressure inside this joint, it would be possible that patients who suffer from knee pain try to decrease their knee flexion as an intuitive mechanism to avoid pain. Less knee flexion angle leading to less amount of patellar movement inward the trochlear groove and consequently, decreased contact with medial and lateral condyles of the femur. This decreased contact pressure may alleviate the soft tissue wear off and irritation over time. However, it may cause an increase in the lower extremity muscle forces and consequently, increase the risk factors of osteoarthritis. More detailed discussion of the topic will be presented in the kinetics section of this chapter.

2.7.3 Stride Length and Linear Velocity of Walking

In a study by Powers et al. (1999) the authors compared the stride length and peak knee flexion angle observed for PFPS subjects to healthy control group in normal and fast walking tasks. Subjects chose their self-desired speed in both normal and fast walking.

Results of the study showed that PFPS patients had a significantly lower velocity of walking and slightly shorter stride length during both fast and normal walking compared to the control group. Moreover, similar to the aforementioned studies regarding joint angles, patients showed a significantly lower peak knee flexion angle during fast walking. Authors suggested that slower walking velocity employed by the PFPS subjects can be a mechanism to minimize the patellofemoral joint reaction force. As a case in point, a slower gait velocity decreases the work demand of the quadriceps muscle group during stance phase by reducing the knee extension moment (Winter. 1984). Additionally, shorter stride length seen among patients in this study can be associated with the lower peak reaction forces observed in patients during fast and normal walking.

2.8 Kinetics Features Associated with PFPS

Main kinetics measures examined in the literature are as follows:

- Ground reaction forces
- Peak joint moments and lower extremity muscle forces

2.8.1 Ground Reaction Forces(GRFs)

As briefly discussed in the previous section, Powers and his colleagues found significantly lower peak values for GRFs during the stance phase of walking in PFPS patients compared to healthy subjects in both fast and normal walking. This might be due to the shorter stride lengths employed by patients in order to decrease the quadriceps muscle

group work demand during the stance phase. In another study, Silva et al. (2015) investigated the differences in peak GRFs, loading rate , and knee flexion angles during stair climbing task between PFPS patients and healthy control group. Similarly, the peak GRFs were significantly lower in PFPS individuals. The loading rate increased, while the knee flexion angles reduced in patients. The decrease in GRFs during stance phase of activities such as walking, running, stair climbing, etc. can be interpreted as a mechanism implemented by patients suffering from anterior knee pain to reduce the foot-ground impact and consequently, decrease the contact forces inside the knee joint.

On the other hand, increased loading rate in patients is claimed to happen due to decreased knee flexion angles. The authors also claim that reducing knee flexion angle during stair climbing in order to avoid anterior knee pain does not seem to be effective to distribute forces applied to lower limb. Higher load peaks acting on the knee in a regular basis can have a detrimental effect. In support of the latter claim, we can mention that increased loading rate may result an enhanced axial compressive force acted on the tibiofemoral joint in a vertical direction. In the literature, this has been addressed to be a contributing factor to initiation of osteoarthritis. However, this increased vertical compressive force may not directly affect the forces applied to patellofemoral joint as it directly dependent on the magnitude of quadriceps muscles force (Silva et al. 2015; Powers et al., 1999). Moreover, decreased knee flexion angle in dynamic tasks implemented by PFPS individuals may reduce active shock absorbing action of the quadriceps muscles (Silva et al. 2015; Cook et al. 1997).

2.8.2 Peak Joint Moments and Lower Extremity Muscle Forces

In a 3D musculoskeletal simulation study by Besier et al. (2009), the authors calculated the knee joint extension moment along with major muscles' forces acting on the lower extremity of the PFPS subjects and healthy persons while walking and running. The results of the study did not show any differences in relative contribution of VM muscle during both walking and running between the groups. During walking, PFPS individuals showed a greater normalized muscle forces compared to control specially during push off phase. However, it was not the case during running as there wasn't any significant difference in normalized muscle forces between the groups. Another finding of the study was lower knee extension moment in PFPS patients compared to control group.

The authors alleged that increased muscle forces can have a detrimental effect of increasing knee joint contact forces. As mentioned earlier, this may increase the risk factors of osteoarthritis. Moreover, the investigators of this study interpreted the increased muscle forces in PFPS patients as a results of increased co-contraction of hamstrings and quadriceps which was seen around heel strike. It is unknown if this increased contact forces are a part of adaptation mechanism by patients to avoid pain or the cause of pain.

In a review study by Weiss and Whatman (2015) similar kinetics and kinematics of patients with PFPS and individuals with history of anterior cruciate ligament (ACL) rupture during different dynamic activities has been investigated. The following figure would be of help

for understanding the terminology and coordinates used in the following section of this chapter.

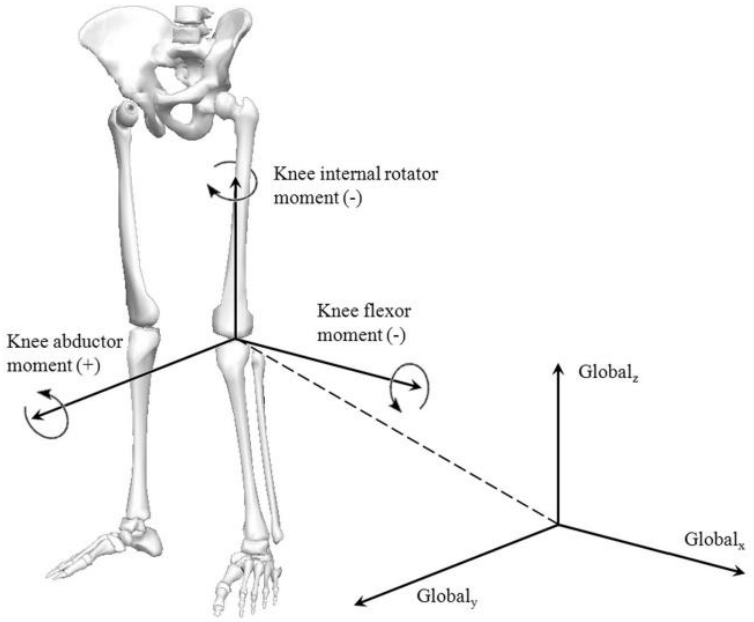

Figure 1. External knee moments with three degrees of freedom [flexor (-x)/extensor (+x), abduction (+y)/adduction (-y), and internal (-z)/ external rotation (+z)] defined locally and with respect to the global coordinate system. Obtained from Weiss and Whatman.

The common variables associated with both ACL injury and PFPS are: Increased knee abduction moment, shallow knee flexion angles, and increased hip flexion angle (because of relative weakness of hamstrings).

Increased knee abduction moment can be a result of increased hip adduction and internal rotation due to weakness of hip musculature. This increased knee abduction moment affects the knee joint structure by stretching the medial collateral (MCL) and ACL ligaments respectively. This also may increase the impact forces acting on the knee joint which increases its injury risk factors.

As discussed earlier, decreased knee flexion angle might be a mechanism in patients to avoid anterior knee pain. However, it claimed that this might not be a proper solution to distribute contact forces applied to the knee and may be a contributing factor to initiate PFPS. Moreover, it has been shown in the literature that during unilateral landing decreased knee flexion angle put large strain force on the ACL due to quadriceps contraction.

Additionally, increased hip flexion angle has been reported in some studies (Weiss and Whatman. 2015). This may be related to weakness of hamstrings muscle group that increases demand on quadriceps muscle group specifically during stance phase and results in increased hip flexion. This may also be interpreted as a mechanism by which the hip joint compensates the decreased knee flexion to dampen the impact and alleviate anterior knee pain during foot contact in various tasks.

2.9 Rehabilitation Methods

As detailed discussion of rehabilitation methods for PFPS is beyond the scope of our investigation in the present study, we shall briefly mention the most common interventions for reduction and treatment of the anterior knee pain.

The major categories of treatment found in the literature are electrical stimulation (ES), physical therapy, patellar taping, muscle strengthening and stretching.

Recently, studies have been focusing on onset timings of the muscles, more specifically GMAX, VL, and VMO. Neuromuscular electrical stimulation (NMES) is widely used to re-educate the firing pattern of a muscle during execution of different tasks.

In a study by Glaviano et al. (2015) the authors found an improved knee flexion and hip abduction kinematics during the lateral step-down exercise in female PFPS patients after a 15-minute patterned NMES of GMED, VMO and hamstrings muscles. Moreover, patients showed an improved GMED activity pattern as well as reduced anterior knee pain. As the study showed encouraging results we may conclude that in PFPS patients who suffer from hip musculature weakness or delayed onset timing of quadriceps, NMES is an effective method for treatment of pain.

In a study by Mason et al. (2011) the authors assessed the effectiveness of quadriceps strengthening, quadriceps stretching and patellar taping and the combined method in a one-week program for PFPS patients. With a combined intervention patients showed the most improvement, strengthening and stretching of quadriceps had similar but less effectiveness than combined method, while patellar taping was the least effective method.

In another study by Crossley et al. (2002), PFPS patients showed significant reduction in their pain scores after a physical therapy program compared to a placebo control group. Treatment program was consisted of retraining and strengthening of quadriceps as well as patellar taping in order to improve the mobilization of the patella.

As identified in the previous sections of this paper, there are various causes for initiation of the anterior knee pain. Consequently, to implement the most effective treatment method it seems necessary to properly diagnose the patient to design a subject-specific

rehabilitation. As an example, if tightness of hamstrings muscle group is the primary cause for a patient's anterior knee pain, electrical stimulation of quadriceps muscles would not be the most effective treatment. Moreover, patellar taping was showed to decrease the pain in patients substantially by correcting the patellar maltracking. And rehabilitation methods have shown to be more effective when the patient does not feel the pain. As a result, usage of patellar taping while performing a combined treatment method for properly diagnosed patients seems to be the most successful rehabilitation method for PFPS patients.

2.10 Conclusion

In this chapter, the mechanism behind patellofemoral pain syndrome (PFPS) was examined from a biomechanical point of view. Maltracking in the movement of the patella was identified as the main cause for PFPS. Accordingly, the biomechanical and neuromuscular factors associated with PFPS due to patellar maltracking were introduced. These factors were reported based on the current scholarly literature available on the topic and were categorized in three groups of static alignments, dynamic malalignments, and abnormal muscle activation. For static alignments, Q-angle and hamstring flexibility had the highest correlation with PFPS. For dynamic malalignments, the results of the studies showed that contralateral pelvic drop, hip excessive internal rotation and adduction, and higher dynamic Q-angle are common patterns in patients diagnosed with PFPS. Weakness of hip musculature was found to be a major contributing factor to the aforementioned patterns. Moreover, higher Q-angles were identified as a causative factor in increased tension of IT band and tightness of VL muscle. They exacerbate the patellar maltracking by

laterally pulling the patella. For abnormal muscle activation, delayed onset timing and relative weakness of VMO muscle to VL muscle was revealed to be prevalent in PFPS patients. Although research studies have not reported any abnormal muscle activation pattern of GMED and GMAX muscles in PFPS patients, strengthening of GMED and GMAX combined with visual feedback was found to decrease the excessive adduction and internal rotation of the hip.

Next, the altered kinematic and kinetic patterns of movement due to PFPS were discussed. Lateral movement of patella, decreased knee flexion angle, increased hip flexion, decreased velocity of walking, and shorter stride length were reported as major altered kinematic patterns observed in PFPS patients compared to healthy individuals. For altered kinetic patterns, decreased GRFs were identified as a mechanism implemented by PFPS patients in order to avoid pain. Furthermore, PFPS subjects showed lower knee extension moment, higher knee abduction moment and higher muscle forces acting on lower extremity than those of the control group. Then, similarities in kinematic and kinetic movement patterns of PFPS patients, individuals with history of ACL rupture and osteoarthritis patients were briefly discussed.

Finally, the most common rehabilitation methods for PFPS such as NMES, physical therapy, patellar taping, and muscle strengthening and stretching were introduced and suggestions were made on the proper design of treatment methods.

CHAPTER 3

METHODS

3.1 Subjects

Twelve volunteers medically diagnosed with PFPS (2 males) aged 19-50 years old participated on this study. Symptomatic PFPS diagnosis on Patella Glide Test, Patella Tilt Test, and Patellar Apprehension Test were implemented as the inclusion criteria. All subjects who had heumatoid arthritis or inflammatory arthritis, ambulation deficiency, diagnosed knee disorder other than PFPS, heart disease, unstable angina, knee replaced in preceding 12 months or replacement planned within 6 months, moderate to severe dementia, pregnancy (self-report), steroid injection within preceding 2 months, and movement-limiting pain in the back, hip, ankle, or foot of either lower limb were excluded from participation.

3.2 Subject Preparation

This study involved one testing session (approximately one hour and half long for each participant) that was approved by the IRB at the University of Texas at Austin. Upon arrival to the Developmental Motor Control Laboratory, the details of all experimental procedures were fully explained to the individual. All participants of the study filled and signed a consent form along with health history questionnaire.

Then, chronic pain level was assessed with the Anterior Knee Pain Scale (AKPS) (Crossley et al. 2004) and the Visual Analog Scale (VAS) (10 cm) (Crossley et al. 2004).

Next, physical assessments such as Patella Glide Test (Testing lateral knee cap mobility), Patella Tilt Test (Testing knee cap mobility by pushing on the lateral side), and Patellar Apprehension Test (Testing for pain following quadriceps contraction) were performed to evaluate if participant's knee pain is related to PFPS.

After it was determined that the participant is eligible to participate in the study, twenty retro-reflective markers were placed on the palpable anatomic landmarks of the legs in preparation for motion capture (Abbas, 2001; Della Croce, 1999). These include femur, tibia, medial & lateral epicondyles, antero-lateral and antero-medial ridges of the patellar surface groove, medial, lateral, proximal, and distal facets of the patella, most medial ridge of the medial tibial plateau, most lateral ridge of the lateral tibial plateau, right and left anterior sacroiliac, right and left ankle, right and left toe, right and left heel. Moreover, wearable bands of microphones model MSI 2-1002785-1 (Articulate Labs, Inc.) were placed over the VL and VMO muscle bellies in order to record the AMG activity of the muscles.

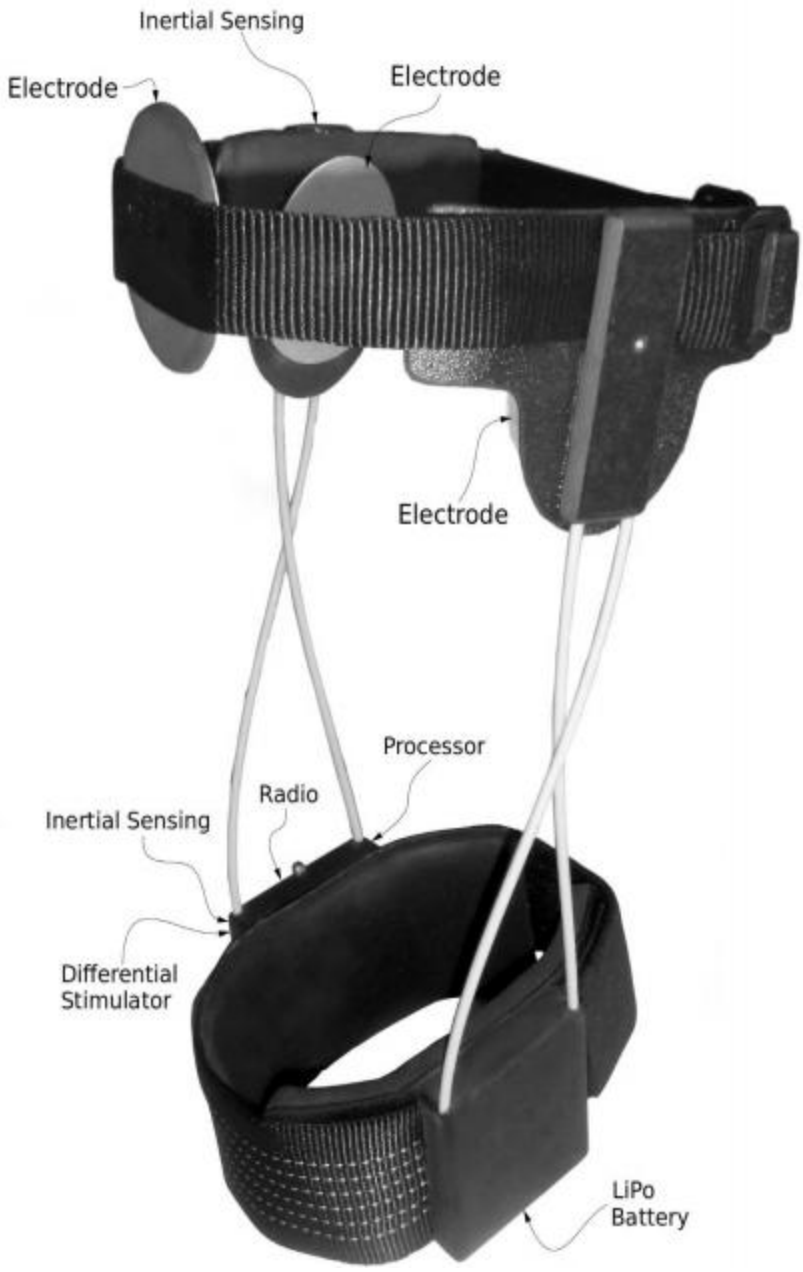

Figure 2. KneeStim Device (Articulate Labs, Inc.)

3.3 Experimental Procedure

Prior to any movement recording, baseline measurements were taken by the VICON motion capture system (with 91.6Hz sampling rate) while the participant was in anatomical standing position. Then, the participant was prepared to complete four periods of 6-minute walking trials with 10-minute resting period in between the walks. Afterwards, to complete the first trial, participants walked back and forth on a straight line in front of the VICON cameras for six minutes (6 Minute Walk Test or 6MWT). Baseline recordings of the participant's knee kinematics and VMO/VL contraction patterns were recorded during this time. After the first 6MWT, a wearable electrical stimulator, KneeStim (Articulate Labs, Inc.), was put on the symptomatic leg (most affected leg by PFPS) of the participant. Following the completion of each 6MWT and during each of the 10-minute rest periods, the AKPS and VAS were re-administered to assess the pain level.

Then participant performed a second 6MWT with the KneeStim device placed on the leg but the stimulation turned off. This was done to better understand what gait and AMG/EMG changes may be caused solely by the physical presence of the device Moreover, during each of the four 6MWTs, the participant was asked to report his/her pain level. This could be an indicator of the points in patient's gait cycle where they experience the most/least pain.

Following the completion of the second 6MWT and during the 10-minute rest period, the intensity of stimulation was determined to help the participant find a muscle stimulation level that is comfortable and elicits quadriceps contraction.
Then, participant performed a third 6MWT with the stimulation. The device was programmed to simultaneously stimulate VMO and VL muscles during the swing phase. Finally, the last 6MWT performed by the participant, with the device being worn without stimulation, in order to observe any changes to the gait and/or VMO/VL contraction patterns originating from the stimulation.

3.4 Data Analysis

3.4.1 Processing of the AMG Signal

The Raw MMG signals were amplified and imported from the analog channels. Then, any offset was removed. Next, the outliers of the signal were detected. Any signal with the amplitude higher than 6 standard deviations of the mean MMG signal was considered as an outlier. These outliers were single high amplitude spikes of the MMG signal that most likely originated when the microphone was disconnected for a moment or sounds originating from heel-strike(HS) events.

A high pass filter with 80Hz cut-off frequency was used to remove the artifacts of HS (Stokes and Dalton, 1991).

The absolute value of the MMG signal was calculated, then the maximum value of each MMG channel was found. To normalize the signal, MMG amplitudes of the both channels (including VL and VMO) were divided by its maximum value respectively.

In order to analyze the frequency domain of the MMG signal, Fast Fourier transform (FFT) can be used (Beck et al., 2005). To examine the patterns of MMG center frequency responses during dynamic tasks the Fast Fourier transform (FFT) was performed (Beck et al., 2006) with Blackman window centered around each data point of the MMG signal.

Window length was determined as exponent of next power of 2 higher than the following:

$$\frac{2 * \text{Sampling rate of the analog data}}{\text{lowest frequency of spectral variance detection}}$$

To examine potential changes in MMG frequency across the range of motion during the dynamic task of walking, a similar method to joint time-frequency was implemented (Beck et al., 2005). Previously high-pass filtered MMG signals of VL and VMO channels were multiplied by their respective normalized spectral variances. MMG onset times were detected during each stride by correlating the tibia acceleration (second derivative of the tibia marker position in time) and the amplitude of the MMG signal.

As the quadriceps firing after toe-off (TO) causes acceleration of the lower leg, the MMG onset times of VL and VM muscles were validated with tibial acceleration in the sagittal plane of motion (measured in body-frame). The force produced (tibia acceleration) by the quadriceps during each stride was identified at the instant that the tibia's acceleration

exceeded 2 standard deviations above its average (instant A) over the full trial. As the muscle contraction occurs slightly earlier than the actual limb movement, the MMG onset times were detected in a backward sliding window. The backward sliding window of 250 milliseconds applied on the MMG signal (ending at instant A) to find the muscle's firing initiating the force (or tibia acceleration). In the sliding window, the instant at which the MMG amplitude exceeded three standard deviations from its baseline was detected as the onset time of the muscle. Finally, the onset times were normalized to the stride period.

It should be mentioned that the MMG onset times were detected only for the symptomatic leg. In order to detect the symptomatic leg, using the four patellar markers' position data, the center of the patella was estimated, then it was correlated with the right or left knee marker. The minimal peak Y-axis error was implemented to detect the leg wearing the KneeStim device.

Finally, the transient (short-duration high amplitude sound noise) was removed from the VL and VMO MMG signals by a 40Hz Brickwall lowpass filter.

3.4.2 Kinematic Events Detection

In order to correlate AMG onset times with its relative kinematic pattern, some of the major gait events such as stride, TO, and HS were measured. To detect the HS events, the instant at which the Z-axis velocity of the ankle marker was less than 0.2 standard deviation of its average velocity was saved as HS event.

In addition, to differentiate TO from HS, the sagittal plane tibial velocity and acceleration were correlated and TO was identified when the product of the velocity and acceleration was positive (Similar signs for acceleration and velocity that is indicative of speeding up). Additional criteria used to detect TO events was the fact that the coronal (vertical) velocity and acceleration become positive and both are increasing at the time of TO.

Finally, the kinematic gait events including the valid TO and HS along with AMG (and EMG) onset times were transferred into a single array with the same sampling rate in order to plot the desired graphs.

3.4.3 Statistical Analysis

A two-way repeated measures analysis of variance (ANOVA) was performed to determine the effects of electrical stimulation and the quadriceps muscle interactions on MMG onset times of the symptomatic leg over the four trials. Following understanding the main effect, pairwise comparison analysis was performed in order to reveal the effect of treatment on the muscle onset times comparing pre and post-stimulation sample means. To investigate the effect of electrical stimulation on difference of VL and VMO muscle onset times before and after treatment, mean stride period and stride length of both non-symptomatic and symptomatic legs, a one-way ANOVA with repeated measures was implemented. A level of significance of $p \leq 0.05$ value was set. All the statistical analyses were done implementing the SPSS software version 24.0.

CHAPTER 4

RESULTS

4.1 Muscle Activation

The onset times of the VMO and VL muscles showed significant change between trial 1 and trial 2, and between trial 2 and 4 (before and after implementation of ES) ($p<0.05$), but no change when trials 1 was compared to trial 4 ($p>0.05$). Figure 3 shows the average onset times of the two muscle over the four trials.

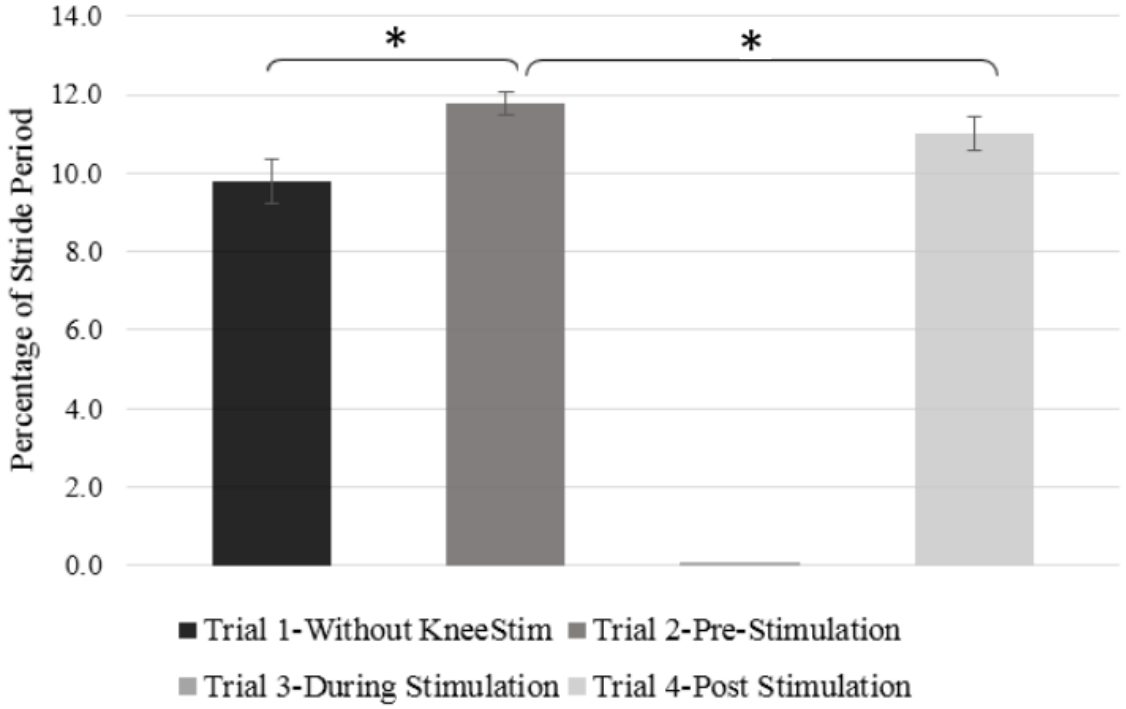

Figure 3. Average muscle onset times of VMO and VL muscles. Muscles showed significantly earlier onset times after treatment. Moreover, the onset time difference of trial 1 Vs. trial 2 was significant. Onset times are normalized based on the stride period starting from toe-off (swing phase initiation). The asterisk indicate statistical significant differences of the sample means, whereas the error bars indicate the standard error of the mean of each sample mean (Trial 1 mean ± S.E 9.8 ± 0.5, Trial 2 mean ± S.E 11.9 ± 0.3, Trial 3 mean ± S.E 0 ± 0, Trial 4 mean ± S.E 10.6 ± 0.3).

The difference of muscles' onset times (VL-VMO) did not change after ES intervention ($p>0.05$). Figure 4 shows the mean value of muscles' onset time difference.

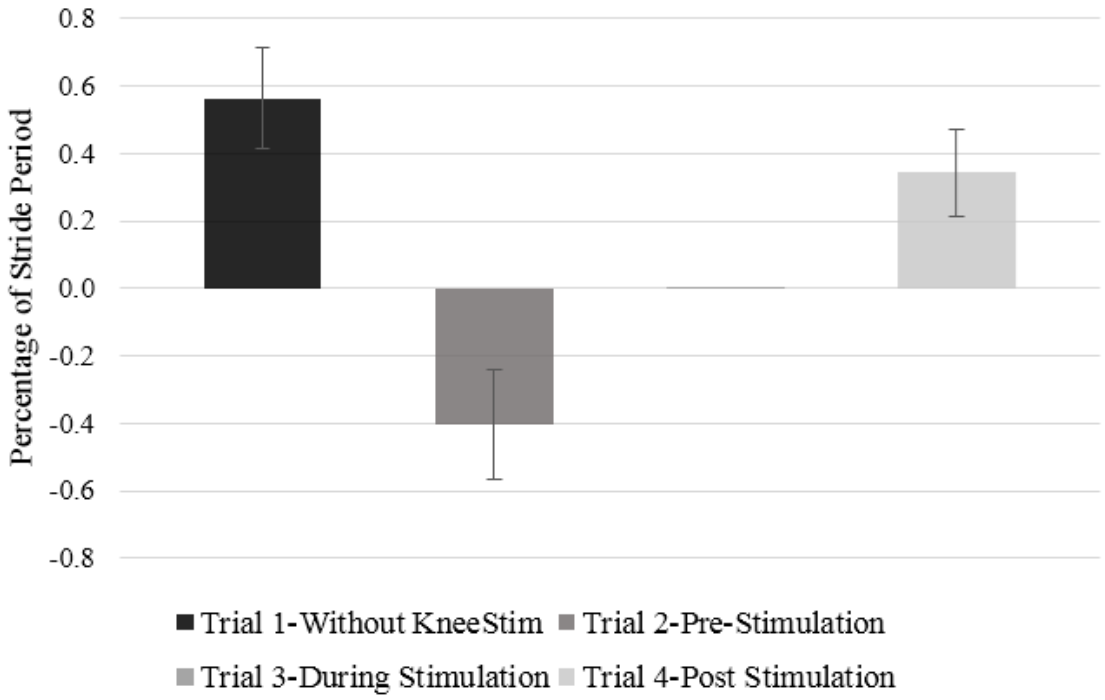

Figure 4. Difference of muscle activation times calculated as VLOnset -VMOOnset. Negative numbers show the delayed activity of VMO compared to VL and positive values show earlier activation of VMO compared to VL. The positive and negative error bars each indicate the half value of the standard error of each sample mean (Trial 1 mean ± S.E 0.6 ± 0.2, Trial 2 mean ± S.E -0.4 ± 0.4, Trial 3 mean ± S.E 0 ± 0, Trial 4 mean ± S.E -0.3 ± 0.3).

The onset times of the VL muscle showed significant change between trial 1 and trial 2, and between trial 2 and 4 (before and after implementation of ES) ($p<0.05$), but no change when trials 1 was compared to trial 4 ($p>0.05$). Figure 5 shows the average onset times of this muscle over the four trials.

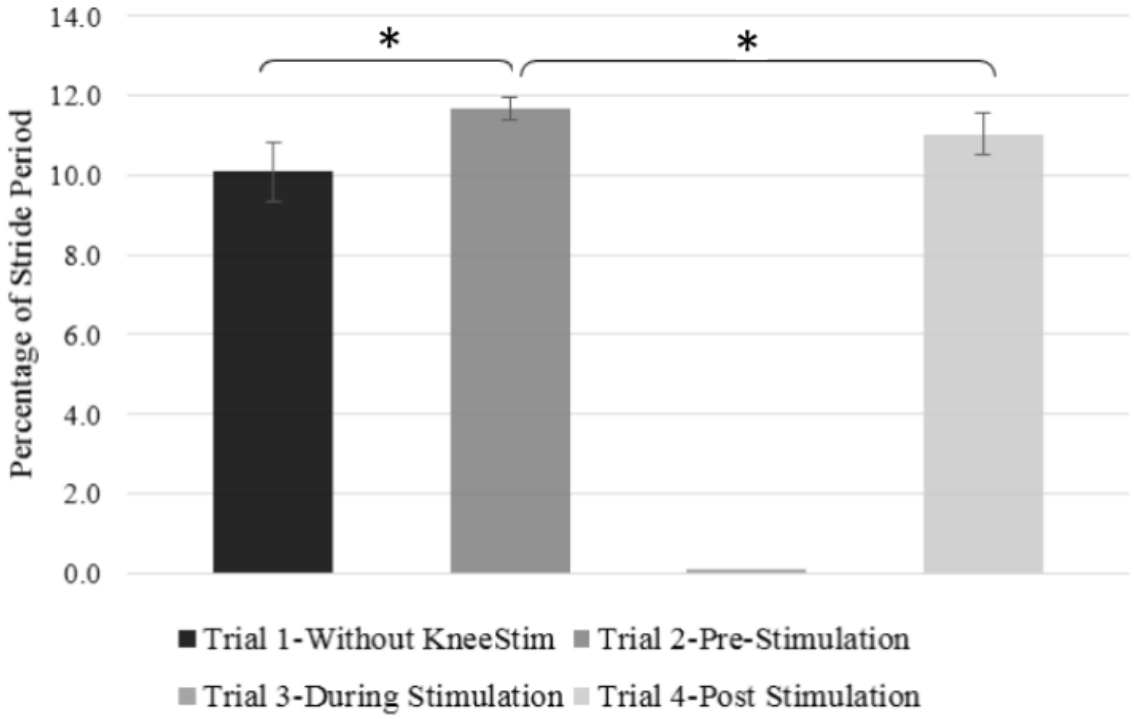

Figure 5. Average muscle onset times of VL muscle during all four trials. The muscle showed significantly earlier onset times after treatment. Moreover, the onset time difference of trial 1 Vs. trial 2 was significant. Onset times are normalized based on the stride period starting from toe-off (swing phase initiation). The asterisk indicate statistical significant differences of the sample means, whereas the error bars indicate the standard error of the mean of each sample mean (Trial 1 mean ± S.E 10.1 ± 0.7, Trial 2 mean ± S.E 11.7 ± 0.3, Trial 3 mean ± S.E 0 ± 0, Trial 4 mean ± S.E 10.7 ± 0.5).

The onset times of the VMO muscle showed significant change between trial 1 and trial 2, and between trial 2 and 4 (before and after implementation of ES) ($p<0.05$), but no change when trials 1 was compared to trial 4 ($p>0.05$). Figure 6 shows the average onset times of this muscle over the four trials.

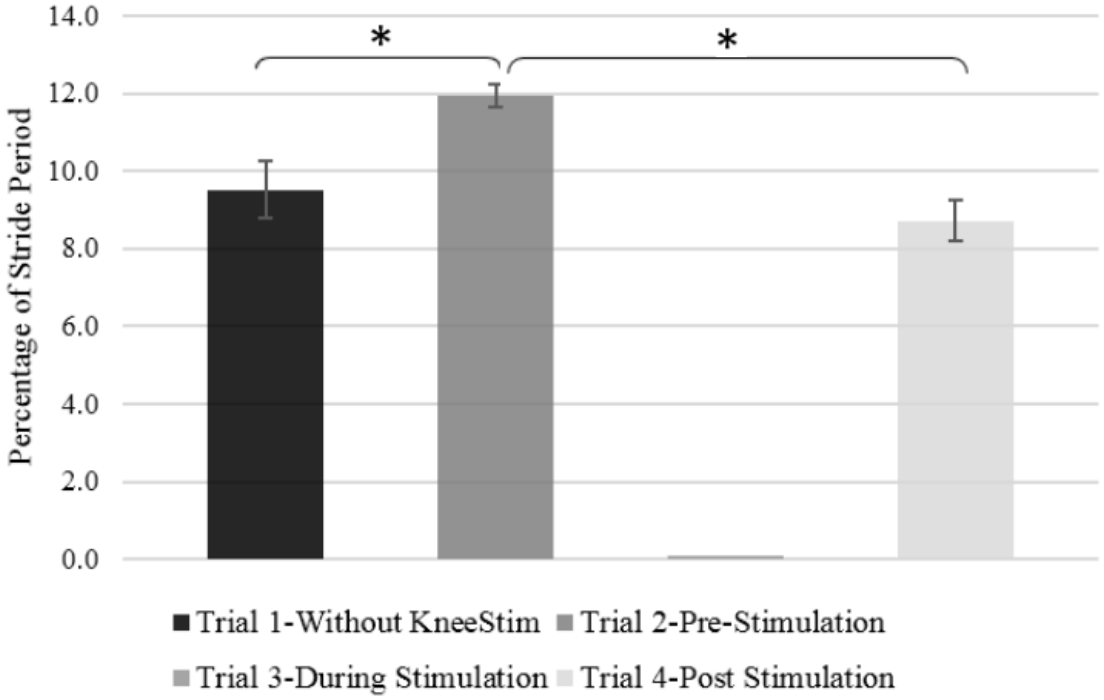

Figure 6. Average muscle onset times of VMO muscle during all four trials. The muscle showed significantly earlier onset times after treatment. Moreover, the onset time difference of trial 1 Vs. trial 2 was significant. Onset times are normalized based on the stride period starting from toe-off (swing phase initiation). The asterisk indicate statistical significant differences of the sample means, whereas the error bars indicate the standard error of the mean of each sample mean (Trial 1 mean ± S.E 9.5 ± 0.8, Trial 2 mean ± S.E 11.9 ± 0.5, Trial 3 mean ± S.E 0 ± 0, Trial 4 mean ± S.E 10.2 ± 0.7).

4.2 Kinematics - Symptomatic Leg Vs. Non-Symptomatic Leg

As shown in figure 7, stride length (mm) values slightly decreased for symptomatic leg after participants were exposed to ES ($p>0.05$). However, this change was significant when non-symptomatic leg was taken into account ($p<0.05$).

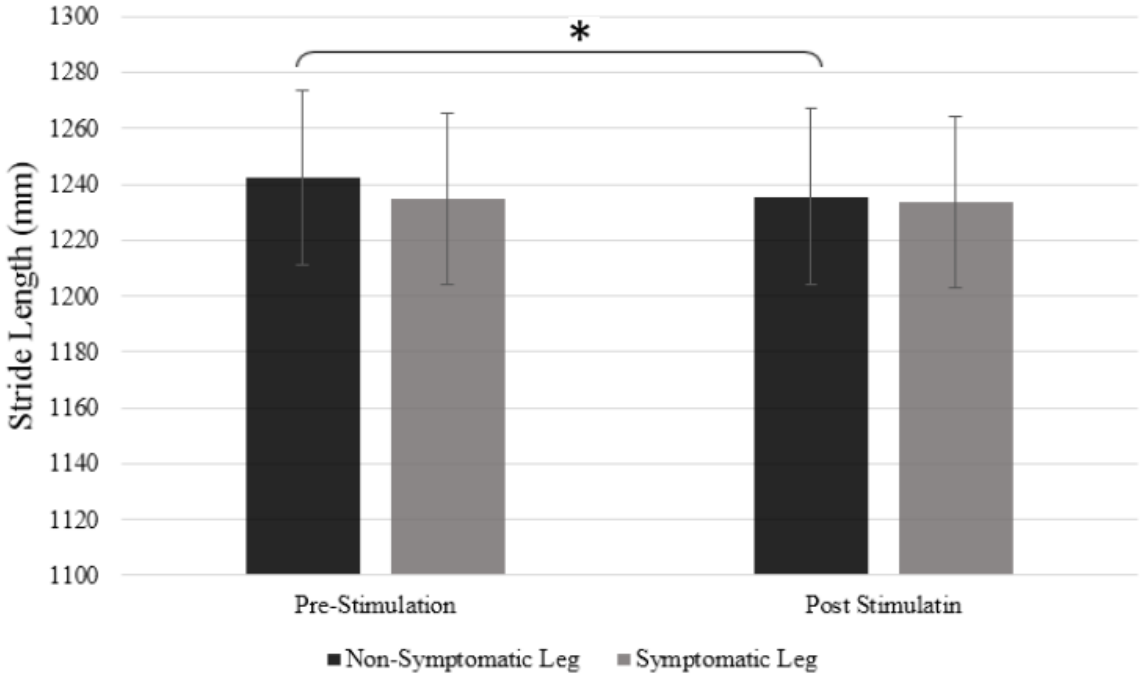

Figure 7. Average stride length (mm) values for symptomatic and non-symptomatic legs of the participants showed insignificant reduction after treatment with ES. The error bars indicate the standard error of the mean of each sample mean (Symptomatic leg: Pre-stimulation mean ± S.E 1234.9mm ± 32.2, post-stimulation mean ± S.E 1233.6mm ± 30.7; Non-Symptomatic leg: pre-stimulation mean ± S.E 1242.3mm ± 31.4, post-stimulation mean ± S.E 1235.5mm ± 31.7).

As shown in figure 8, stride period (ms) did not change after exposure to ES for both symptomatic and non-symptomatic legs ($p<0.05$).

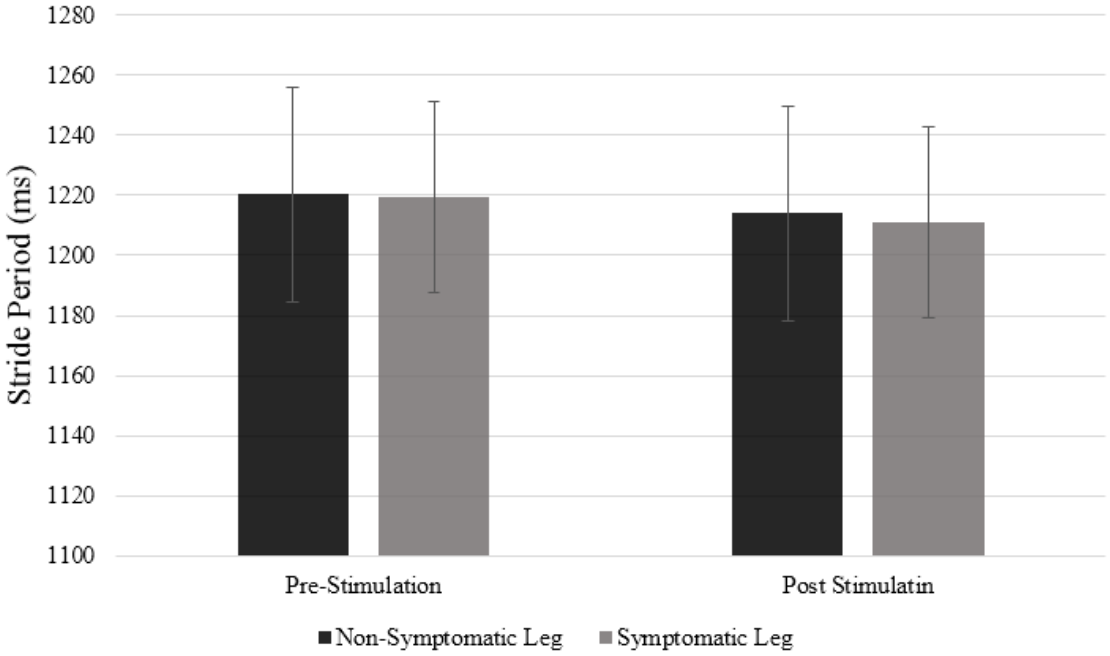

Figure 8. Average stride period (ms) values for symptomatic and non-symptomatic legs of the participants showed insignificant reduction after treatment with ES. The error bars indicate the standard error of the mean of each sample mean (Symptomatic leg: pre-stimulation mean ± S.E 1219.2ms ± 35.1, post-stimulation mean ± S.E 1211.1ms ± 31.7; Non-Symptomatic leg: pre-stimulation mean ± S.E 1220.3ms ± 35.7, post-stimulation mean ± S.E 1213.9ms ± 33.8).

CHAPTER 5

DISCUSSION

The purpose of this study was to examine the short-term effects of ES on quadriceps muscle activation pattern and gait cycle kinematics during normal walking in individuals with PFPS. The primary hypothesis of the study was that following synchronous ES of VL and VMO muscles at the beginning of each stride during trial 3 of the experiment, PFPS patients would show an earlier activation onset times of these muscles. Second hypothesis of the study was that the onset time difference of VL and VMO muscles would decrease following the implementation of ES. Finally, it was hypothesized that patients with PFPS would show an increase in stride length after intervention.

The results of our study confirmed that ES treatment led to earlier onset times of VL and VMO muscles significantly. However, the onset time difference of VL and VMO did not change.

Abnormal muscle activation pattern in PFPS has been divided in to two main categories; Onset timing of the muscles and the intensity of muscle activation which both can lead to maltracking of the patella (Guney et al., 2016).

Miller and colleagues (1997) examined the relative strength of the VMO and VL muscles in PFPS patients while performing closed kinetic chain exercises. The PFPS group showed less EMG activity of VMO compared to VL. This resulted in less relative ratio of

VMO:VL activity in individuals with PFPS compared to the healthy control group. Weakness of the VMO has been associated with pain as it causes an imbalance in lateral/medial forces acting on the patella and consequently, pulling the patella more laterally in trochlear groove of the femur during movement (Sawatsky et al., 2011; Sakai et al., 2000). This causes overload on the lateral side of the patellofemoral joint which can be resulted in pain (Dhaher and Khan, 2002).

On the contrary, a recent study (Toumi et al., 2013) alleged that PFPS is not necessarily associated with quadriceps deficit. During squat and isometric strength test maneuver, they found delayed onset of VMO muscle (VMO activation deficit) in 17 PFPS subjects and observed quadriceps strength deficit in only 6 PFPS subjects out of 32 patients recruited for the research study. Similarly, in our study and during trial 2 (pre-stimulation), 6 participants showed delayed activation of VMO compared to VL. This delay on average for participants of our study was 0.4% when compared to VL.

In terms of rehabilitation, the most common interventions for reduction and treatment of the anterior knee pain are electrical stimulation (ES), physical therapy, patellar taping, muscle strengthening and stretching.

Recently, studies have been focusing on onset timings of the muscles, more specifically VL and VMO as the main cause of anterior knee pain initiation. In a study by Glaviano et al. (2015) the authors found an improved knee flexion and hip abduction kinematics during the lateral step-down exercise. This was obtained in female PFPS patients after a 15-minute patterned neuromuscular electrical stimulation (NMES) of gluteus medius (GMED), VMO and hamstrings muscles.

Additionally, in a study by Crossley et al. (2002), PFPS patients showed significant reduction in their pain level after a treatment program consisting of quadriceps strengthening as well as patellar taping. As a result, our hypothesis was that in PFPS patients who suffer from hip musculature weakness or delayed onset timing of quadriceps, electrical stimulation is an effective method for treatment of pain. Likewise, the results of our study showed delayed onset of VMO compared to VL during the dynamic task of walking before intervention. However, after short term ES intervention, VMO showed earlier activation but the difference of muscles' onset times did not change. On the other hand, when the effect of the KneeStim was taken into account, the average muscle onset times during trial 1 was significantly lower than trial 2. This shows that the KneeStim device without being activated solely caused later activation of the quadriceps muscle. This could be addressed by the fact that the device could solely cause increase in knee flexion angle, preventing the knee joint to be fully extended at the beginning of each stride. Later extension of the knee joint following increased knee flexion angle can cause later activation of VL and VMO compared to trial 1 when the KneeStim device was not installed.

In contrast to our primary and secondary hypotheses, the results of our study was inconsistent with our final hypothesis as the stride length decreased following the ES intervention.

Powers et al. (1999) have reported shorter stride length in PFPS individuals during normal walking when compared to the healthy control group. The authors compared the stride length and peak knee flexion angle observed in PFPS subjects to the

healthy control group in normal and fast walking tasks. Subjects chose their self-desired speed in both normal and fast walking. Results of the study showed that PFPS patients had a significantly lower velocity of walking and slightly shorter stride length during both fast and normal walking compared to the healthy control group. Similarly, it was assumed that ES would improve the tracking of the patella in PFPS patients by assisting VL and VMO muscles to synchronously activate and consequently decrease the pain level. As a result, it was expected to observe an increase in stride length. This assumption was made based on the fact that the shorter stride length in PFPS patients could be due to the knee pain initiating from patella maltracking (Haim et al., 2006 and Baker et al., 2002) and consciously avoiding the full knee extension and high knee flexion angles while walking. This strategy implemented by patients could be due to the quadriceps deficiency which may cause an increase in patellofemoral joint's contact pressure during the full knee extension and flexion. This idea was supported by a simulation study in which the authors reported that strengthening of VL muscle can lead to significant reduction of patellofemoral contact force and diminution of contact area at 90 degree knee flexion (Wünschel et al., 2011). Moreover, decreased knee-extension moment during different tasks such as walking and stair climbing in PFPS patients has been reported (Salsich et al., 2001; Heino and Powers, 2002). This was explained as a strategy implemented by PFPS patients in which the participants of the study lowered their gait velocity to avoid pain due to elevated knee-extension moment.

Consequently, expecting longer stride length after ES intervention would be reasonable. However, in our study the knee flexion/extension angles and moments were

not calculated to increase the accuracy of monitoring the changes in participants' pattern of movement due to ES. Additionally, one could also argue that a 6-minute ES intervention would be too short to make any major changes in gait pattern. Moreover, the participants of our study did not report any major pain during the experimental session even during the intervention. This could also explain why our participants did not show any major improvement on their measured gait kinematics.

To our knowledge, no research study has been conducted to assess the effects of ES on stride period. In our study, as participants were asked to maintain the same walking speed by following the metronome signal during four walking trials, no major changes were expected on stride period after intervention. The small reduction observed in stride period was not considerable.

Although our results were mostly expected and favorable, there are limitations that could be addressed in future research. First, the small number of participants (12), that could explain why ES intervention did not show significant reduction of quadriceps muscle onset time difference compared to pre-stimulation. Second, the intervention involved only a single 6-minute treatment. Longer periods of stimulation with the KneeStim device (a unique and more convenient method) should be examined. As the results of our ES intervention were encouraging, it would be interesting to design a rehabilitation program with the KneeStim device in future research.

Third, pain reported by the participants of the study was on the low end of both AKPS and VAS scales. It is not clear whether individuals with more severe signs of PFPS would also have the same outcome.

Finally, normal walking might not be the best indicator of VMO and VL function as the activation level of these muscles are relatively low. More demanding tasks such as squatting, stair climbing, etc. could be more illustrative when assessing the quadriceps muscle activation levels.

CHAPTER 6

CONCLUSION

A single session ES intervention starting at the beginning of each stride showed earlier activation of quadriceps muscles during normal walking in individuals with PFPS. However, the delay onset timing of VL compared to VMO did not change after treatment. Future research should focus on a long-term rehabilitation method for PFPS population implementing ES with more accurate pain and gait kinematic assessments while using a variety of dynamic tasks.

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